UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Ex parte KUO-FEN LEE,
WYLIE W. VALE, TRACY L. BALE,
and GEORGE W. SMITH

Appeal No. 2006-0485
Application No. 09/714,692

ON BRIEF

Before ELLIS, SCHEINER and GRIMES, Administrative Patent Judges.

GRIMES, Administrative Patent Judge.

DECISION ON APPEAL

This appeal involves claims to a method of inhibiting angiogenesis, which the examiner has rejected as anticipated by the prior art. We have jurisdiction under 35 U.S.C. § 134. We affirm.

Background

The specification discloses that "angiogenesis may be inhibited in a target tissue by administering a [Corticotropin Releasing Factor Receptor 2] CRFR2 agonist such as urocortin or CRF." Page 8, lines 6-8. The specification discloses that such target tissues

include "heart, brain, pituitary, gonad, kidney, adipose, or gastrointestinal tract tissues." Page 25, line 21 – page 26, line 1. In addition, "CRFR2 agonist-induced inhibition of angiogenesis may be used in the treatment of cancer." Page 8, lines 7-10.

Discussion

1. Claim construction

Claims 20-23 are pending. Since Appellants have not argued the claims separately, they will stand or fall together. See 37 CFR 41.37(c)(1)(vii). We will focus on claim 20, the only independent claim on appeal, which reads:

> 20. A method of inhibiting angiogenesis in a target tissue comprising the step of administering a Corticotropin Releasing Factor Receptor 2 (CRFR2) agonist to said target tissue, wherein said CRFR2 agonist inhibits angiogenesis in said tissue.

Thus, claim 1 is directed to a method of administering a CRFR2 agonist, e.g., a CRF, to a target tissue, e.g., brain, to inhibit angiogenesis in said tissue.

2. Anticipation

The examiner rejected claims 20-23 as anticipated by Villalona-Calero.[1] Villalona-Calero teaches administering a CRFR2 agonist, specifically human corticotropin releasing factor (hCRF), to "inhibit[] vascular leakage of plasma." Page 71, column 2, last paragraph. More specifically, "hCRF reduces water content in tumor and peritumoral tissue in brain tumor models in vivo when administered subcutaneously." Page 76,

[1] Villalona-Calero et al., "A phase I trial of human corticotropin-releasing factor (hCRF) in patients with peritumoral brain edema," Ann. Onc., Vol. 9, pp. 71-77 (1998).

column 1, first paragraph. Villalona-Calero also teaches that this "effect...[is] a direct action on the tumor microvasculature." Page 76, column 1, first paragraph. Consequently, the examiner has taken the position that inhibition of angiogenesis must be inherent in Villalona-Calero because the CRFR2 agonist, i.e., hCRF, is administered to the same subject population and same tissue as recited in the instant claims. See the Examiner's Answer, page 3. We agree that the disclosed method meets all the limitations of claim 20.

Appellants argue that "Villalona-Calero et al. teach human corticotropin releasing factor inhibits vascular leakage of plasma constituents in response to injury (last sentence on page 71)...[and] do not teach or suggest a method of using corticotropin releasing factor to inhibit angiogenesis in a target tissue as claimed." Appeal Brief, page 7. More specifically, Appellants assert that "the Examiner has not provided any basis in fact and/or technical reasoning to reasonably support the legal determination that the allegedly inherent characteristic of inhibiting angiogenesis necessarily flows from the teaching of Villalona-Calero." Appeal Brief, page 9. Appellants conclude that "[a]bsent a teaching that shows any relationship between angiogenesis and anti-edematous effects, one of ordinary skill in the art would have no reasonable and logical scientific basis to recognize or suspect that human corticotropin releasing factor inherently possesses angiogenesis-inhibiting activity." Appeal Brief, page 7. Thus, "Appellants contend that the Examiner has failed to make out a prima facie anticipation, and has failed to provide a reasoned explanation of how a discussion of inhibition of vascular leakage of plasma constituents or actions on brain tumor microvasculature in Villalona-Calero et al. would

lead one with skill in the art to the conclusion that human CRF inhibits angiogenesis in brain tissue." Reply Brief, page 2.

This argument, however, is not persuasive. First, it is well known that "[a] claim is anticipated only if each and every element as set forth in the claim is found, either expressly or inherently described, in a single prior art reference." Verdegaal Bros., Inc. v. Union Oil Co., 814 F.2d 628, 631, 2 USPQ2d 1051, 1053 (Fed. Cir. 1987) (emphasis added). "'[I]t is elementary that the mere recitation of a newly discovered function or property, inherently possessed by things in the prior art, does not cause a claim drawn to those things to distinguish over the prior art. Additionally, where the Patent Office has reason to believe that a functional limitation asserted to be critical for establishing novelty in the claimed subject matter may, in fact, be an inherent characteristic of the prior art, it possesses the authority to require the applicant to prove that the subject matter shown to be in the prior art does not possess the characteristic relied on.'" In re Best, 562 F.2d 1252, 1255, 195 USPQ 430, 433 (CCPA 1977) (quoting In re Swinehart, 439 F.2d 210, 212-13, 169 USPQ 226, 229 (CCPA 1971)).

In this case, Villalona-Calero teaches that hCRF (a CRFR2 agonist) "reduces water content in tumor and peritumoral tissue in brain tumor models in vivo when administered subcutaneously...[and] [t]his effect [is] a direct action on the tumor microvasculature." Page 76, column 1, first paragraph. Additionally, "the broad method steps claimed in the instant application are the same steps disclosed in Villalona-Calero." Examiner's Answer, page 6. Since the CRFR2 agonist is administered to the same subject population and same tissue, e.g., administering hCRF to patients with brain tumors, as those recited in Appellants' specification and claims, it would be expected to

inherently result in angiogenesis inhibition, even if that effect was not recognized. Furthermore, "[f]rom the standpoint of patent law, a compound and all of its properties are inseparable; they are one and the same thing." In re Papesch, 315 F.2d 381, 391, 137 USPQ 43, 51 (CCPA 1963). Thus, stating a new property of CRF does not distinguish the claimed method of inhibiting angiogenesis from the inherent anticipation of the prior art. See Examiner's Answer, page 7. As a result, the examiner has established a prima facie case of anticipation.

"[A]fter the [examiner] establishes a prima facie case of anticipation based on inherency, the burden shifts to appellant to 'prove that the subject matter shown to be in the prior art does not possess the characteristic relied on.'" In re King, 801 F.2d 1324, 1327, 231 USPQ 136, 138 (Fed. Cir. 1986) (quoting In re Swinehart). In this case, Appellants' argument does not establish a difference between the claimed invention and prior art method. Nothing in the specification or claims provides any details regarding the effective amount of CRFR2 for administration or in any other way distinguishes the claimed method from the prior art. Appellants' specification merely discloses that results from "anti-PECAM immunostaining...confirm[] that the absence of the CRFR2 receptor in the null mutant mice results in an increase in number and size of blood vessels," which is the basis for the conclusion that "one of the roles of the CRFR2 receptor...is to mediate a CRF-induced inhibition of angiogenesis." Page 50, lines 9-18 (emphasis added). As a result, Appellants have failed to satisfy the burden to disprove inherency.

Appellants also argue that, under In re Marshall, 578 F.2d 301, 198 USPQ 344 (CCPA 1978), even "if a claimed method comprises steps identical to those of a method practiced in the prior art, and the same result would have been achieved in the prior art

method, the 'accidental or unwitting achievement of that result cannot constitute anticipation.'" Reply Brief, page 3.

This argument is also unconvincing. The facts of this case are distinguishable from Marshall. Villalona-Calero and the instant claims are both directed to the same subject population and same tissue, e.g., administering CRFR2 in patients with brain tumors, whereas in Marshall, the claims were directed to a weight control process and the cited reference was directed to treatment of esophagitis, gastritis, peptic ulcer and irritable colon syndrome, and the anesthetic release of gastrin. Reply Brief, page 3.

Even assuming, for the sake of argument, that Appellants were the first discover that administering hCRF to brain tissue inhibited angiogenesis, "[i]t is a general rule that merely discovering and claiming a new benefit of an old process cannot render the process again patentable." In re Woodruff, 919 F. 2d 1575, 1578, 16 USPQ2d 1934, 1936 (Fed. Cir. 1990). "Newly discovered results of known processes directed to the same purpose are not patentable because such results are inherent." Bristol-Myers Squibb Co. v. Ben Venue Labs., Inc., 246 F.3d 1368, 1376, 58 USPQ2d 1508, 1514 (Fed. Cir. 2001). For the reasons stated above, Appellants have failed to provide and establish sufficient evidence to show that the method disclosed by Villalona-Calero does not meet all the limitations of instant claim 20. Thus, we agree with the examiner that Villalona-Calero is anticipatory.

Claim 20 reads on the method disclosed by Villalona-Calero and is therefore unpatentable. Claims 21-23 fall with claim 20. The examiner's rejection is affirmed.

No time period for taking any subsequent action in connection with this appeal may be extended under 37 CFR § 1.136(a).

AFFIRMED

Joan Ellis
Administrative Patent Judge)
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Toni R. Scheiner) BOARD OF PATENT
Administrative Patent Judge)
) APPEALS AND
)
) INTERFERENCES
)
Eric Grimes)
Administrative Patent Judge)

EG/jlb

David L. Parker
Fulbright & Jaworski LLP
600 Congress Avenue
Suite 2400
Austin, TX 78701